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Volume 64, Issue 4, 22 February 1991, Pages 693-702

doi:10.1016/0092-8674(91)90499-O Cite or Link Using DOI
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Article

Targeted disruption of the c-src proto-oncogene leads to osteopetrosis in micePhilipps Soriano^{b, a}, Charles Montgomery^c, Robert Geske^c and Allan Bradley^c^a Howard Hughes Medical Institute Baylor College of Medicine, Houston, Texas 77030, USA^b Institute for Molecular Genetics Baylor College of Medicine, Houston, Texas 77030, USA^c Center for Comparative Medicine Baylor College of Medicine, Houston, Texas 77030, USA

Received 20 November 1990. Available online 25 August 2004.

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Abstract

To understand the normal, physiological role of the *c-src* proto-oncogene, a null mutation was introduced into the gene by homologous recombination in mouse embryonic stem cells. Two independent targeted clones were used to generate chimeras that transmitted the mutated allele to their offspring. Intercrossing of heterozygotes gave rise to live born homozygotes, but most of these mice died within the first few weeks of birth. Histological and hematological examination of the homozygous mutants did not reveal detectable abnormalities in the brain or platelets, where *src* is most highly expressed. However, these mutants were deficient in bone remodelling, indicating impaired osteoclast function, and

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developed osteopetrosis. These results demonstrate that *src* is not required for general cell viability (possibly because of functional overlap with other tyrosine kinases related to *src*) and uncover an essential role for *src* in bone formation.

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